
SHAPING FUTURES: PROGNOSIS, RECOVERY, AND THE VITAL ROLE OF REHABILITATION IN POST-CARDIAC ARREST CARE

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Recent advances in cardiopulmonary resuscitation such as targeted temperature management, and intensive care protocols have significantly improved survival rates following cardiac arrest. However, with increasing survival comes a parallel rise in cases of hypoxic-ischemic brain injury, a leading cause of morbidity and long-term disability in these patients. This presentation aims to provide a comprehensive overview of the pathophysiology, clinical manifestations, prognostication challenges, and rehabilitation approaches associated with brain injury after cardiac arrest. Cerebral ischemia during cardiac arrest leads to a cascade of metabolic disturbances. Within seconds of circulatory arrest, oxygen depletion impairs neuronal function, and within minutes, glucose is exhausted, resulting in the failure of ATP-dependent ion pumps. This dysfunction promotes massive influxes of calcium and the release of glutamate, which in turn activates NMDA receptors, exacerbating excitotoxic injury.

The intracellular calcium burden triggers mitochondrial dysfunction and the production of reactive oxygen species, initiating cell death pathways. Secondary injury processes—such as cerebral edema, impaired autoregulation, and hypotension—can occur hours to days later, further amplifying neuronal damage. Clinically, the brain regions most vulnerable to hypoxia include the hippocampus, thalamus, cerebral cortex, and basal ganglia. Neurological sequelae vary widely, ranging from subtle memory and executive function impairments to coma and persistent disorders of consciousness. Movement disorders (e.g., myoclonus, Parkinsonism), seizures, paroxysmal autonomic instability and spasticity may also develop, complicating both acute management and long-term recovery. Prognostication in this population remains challenging, particularly in the context of therapeutic hypothermia, which may mask clinical signs due to sedation and delayed metabolism of medications.

Current recommendations suggest waiting at least 72 hours after achieving normothermia to perform neurological assessment, including brainstem reflex testing, EEG, and neuroimaging. Early signs such as myoclonic status epilepticus or absent brainstem reflexes may indicate poor prognosis, but must be interpreted with caution. Despite early pessimism, a proportion of patients recover meaningful function, emphasizing the importance of delayed, multimodal prognostication. Scales such as the Glasgow Outcome Scale (GOS) and the Cerebral Performance Category (CPC) remain standard, though their predictive value can be affected by early withdrawal of care. Rehabilitation is a cornerstone of management in survivors after the cardiac arrest. A tailored, interdisciplinary approach—initiated as early as clinically feasible—should address motor deficits, cognitive dysfunction, emotional dysregulation, and reintegration into daily life.

Functional neurorehabilitation should be guided by structured assessment tools and include intensive physical therapy, speech and language therapy, neuropsychological interventions, and caregiver support. From a systems perspective, integrating rehabilitation planning into the continuum of care—from ICU to outpatient follow-up—is essential. Developing standardized post-cardiac arrest pathways with early involvement of rehabilitation specialists improves functional outcomes and reduces long-term dependency. In conclusion, as survival after cardiac arrest improves, the medical community must address the complexity of neurological outcomes and the critical role of rehabilitation. Understanding the underlying pathophysiological mechanisms of brain injury, utilizing cautious and evidence-based prognostication, and implementing early, individualized rehabilitation strategies are essential to optimizing recovery and quality of life in this growing patient population.

Keywords: Brain injury, anoxia, encephalopathy, cardiac arrest

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